

EXHIBIT J

Expert Report of Dr. Michael A. Mont
In re Bair Hugger Forced Air Warming Devices Products Liability Litigation

June 1, 2017

Corey L. Gordon, Esquire
Blackwell Burke P.A.
431 So. 7th St. Suite 2500
Minneapolis, Minnesota 55415

Re: **Bair Hugger Forced Air Warming
Products Liability Litigation**

Dear Mr. Gordon:

At your request, I have prepared this summary of the expert opinions on the issue of general causation I plan to offer in the Bair Hugger multidistrict litigation in federal court in Minnesota.

References and reliance materials upon which I base my opinion are listed in Exhibit A, attached hereto. In addition, I rely upon my background, training, experience and research. My CV, including a list of publications, is attached hereto as Exhibit B. I am being compensated at the rate of \$500 per hour for my preparation of this report and testimony. A list of cases in which I have testified as an expert witness is attached hereto as Exhibit C.

MY BACKGROUND

I am an orthopaedic surgeon, board certified by the American Academy of Orthopaedic Surgeons (AAOS). I received my medical degree from the University of Pennsylvania in 1984. From 1984 to 1989, I did a research fellowship, followed by an internship, and then an orthopaedic residency at the Mt. Sinai Medical Center, Department of Orthopaedics, in New York City. From 1989 to 1990, I completed a one-year fellowship in lower extremity joint reconstruction at the Johns Hopkins University Medical Institutions, Department of Orthopaedics

in Baltimore, Maryland. Following this, I stayed on the full-time orthopaedic faculty at Hopkins from 1990 to 2000 as an Assistant and then as an Associate Professor of Orthopaedic Surgery. In 2000, I co-founded the Rubin Institute for Advanced Orthopedics at Sinai Hospital of Baltimore and became the Director of the Center for Joint Preservation and Replacement. I held this position through June of 2017 and had become an Adjunct Associate Professor of Orthopaedic Surgery at Hopkins. As of July 2017, I assumed my present position as Chairman of Orthopaedic Surgery at the Cleveland Clinic, Cleveland, Ohio.

I routinely take care of lower extremity joint arthroplasty patients. I have performed during my professional career over 500 to 700 joint replacement surgeries per year for a total of over 15,000 since 1990. I have typically seen over 6,000 patients per year with approximately half of them being related to knee arthroplasties (although with duties as Chairman this past year my clinical activity has been reduced).

I have been course director of many local, national, and international meetings that deal with hip and knee replacement surgery. Through these meetings, and my training and experience, I know the standard of care for treating patients with multiple medical and surgical issues, including known complications from surgical procedures such as periprosthetic infections (PJIs). This knowledge is garnered not only from my fellow panelists, but also from general orthopaedists in the audience when we go over case reports of patients who have similar issues to the ones presented in this litigation.

I am a member of the American Association of Hip and Knee Surgeons (AAHKS), the Hip Society, the Knee Society, and the International Hip Society. I am on the editorial board of over ten different journals including being the Associate Editor of the *Journal of Arthroplasty*. I have received numerous grants related to knee and hip arthroplasty (greater than 100).

Concerning the topic of lower extremity joint arthroplasty, I have close to 700 peer-reviewed PubMed publications. Many of these are related to the topic of periprosthetic infections.

OPINIONS

The major source of periprosthetic joint infections (PJIs) is the patient's own skin.

It is well-established that the major source of bacteria causing PJIs is the patient's own body, particularly the skin. This is why so many of the preventative efforts in the operating room are directed in this arena. Bacteria exist everywhere on the human body -- in our skin, our gut, etc.-- it is commonly believed that bacteria outnumber the cells in our body by as much as 10 to 1.

A brief discussion about prevention of PJIs is in order to put the above statement into appropriate context as well as to understand a number of points that will be made later on in this report. The factors that can cause PJIs involve the host and the environment. Host factors include, but are not limited to, smoking, alcoholism, uncontrolled diabetes, renal failure, rheumatoid arthritis, history of infections, and malnutrition, among others. Some of these are modifiable or optimizable before surgery and some are not. These host factors can have an enormous impact on the patient's own bacterial bioburden, as well as the patient's ability to resist infection from endogenous bacteria (bacteria that come from the patient). These host factors can also diminish the ability of the patient to avoid infection from exogenous sources of bacteria (external to the patient) as well. As discussed herein, exogenous sources of bacteria are responsible for a small minority of PJIs.

Preoperative strategies to reduce the risk of PJIs include skin cleansing (bacterial decolonization), and prophylactic antibiotics. Intra-operative strategies include hair clipping (not shaving), skin preparation, surgeon and staff preparation, gloving and gowning, draping, antibiotics in the cement, blood conservation, and overall surgical techniques, including copious wound irrigation.

The operating room environment has a multitude of sources of potential contamination. This should be minimized, as much as possible, by not prolonging surgeries unnecessarily to minimize further skin or wound contamination, minimizing operating room traffic, and being careful about contamination of necessary equipment, e.g. suction tips, blades, saws, light handles, etc. Post-operatively antibiotics are often given and appropriate wound aftercare is administered. Patients may need prophylactic antibiotics before certain medical, dental and/or surgical procedures.

I have conducted substantial research concerning the prevention of PJIs with skin preparations, most notably chlorhexidine. We have shown that advance skin preparation can reduce the incidence of PJIs by 60-70 % or greater. The following is a partial list of my publications in this important arena for infection prevention:

1. Chlorhexidine reduces infections in knee arthroplasty. Johnson AJ, Kapadia BH, Daley JA, Molina CB, Mont MA. J Knee Surg. 2013 Jun;26(3):213-8.
2. Economic evaluation of chlorhexidine cloths on healthcare costs due to surgical site infections following total knee arthroplasty. Kapadia BH, Johnson AJ, Issa K, Mont MA. J Arthroplasty. 2013 Aug;28(7):1061-5.
3. Pre-admission cutaneous chlorhexidine preparation reduces surgical site infections in total hip arthroplasty. Kapadia BH, Johnson AJ, Daley JA, Issa K, Mont MA. J Arthroplasty. 2013 Mar;28(3):490-3.
4. Does Preadmission Cutaneous Chlorhexidine Preparation Reduce Surgical Site Infections After Total Knee Arthroplasty? Kapadia BH, Zhou PL, Jauregui JJ, Mont MA. Clin Orthop Relat Res. 2016 Jul;474(7):1592-8.
5. Preoperative skin disinfection methodologies for reducing prosthetic joint infections. Banerjee S, Kapadia BH, Mont MA. J Knee Surg. 2014 Aug;27(4):283-8.
6. Does Preadmission Cutaneous Chlorhexidine Preparation Reduce Surgical Site Infections After Total Hip Arthroplasty? Kapadia BH, Jauregui JJ, Murray DP, Mont MA. Clin Orthop Relat Res. 2016 Jul;474(7):1583-8.
7. Preoperative chlorhexidine preparation and the incidence of surgical site infections after hip arthroplasty. Johnson AJ, Daley JA, Zywiell MG, Delanois RE, Mont MA. J Arthroplasty. 2010 Sep;25(6 Suppl):98-102

8. Patient Compliance with Preoperative Disinfection Protocols for Lower Extremity Total Joint Arthroplasty. Kapadia BH, Cherian JJ, Issa K, Jagannathan S, Daley JA, Mont MA. Surg Technol Int. 2015 May;26:351-4.
9. Advance pre-operative chlorhexidine reduces the incidence of surgical site infections in knee arthroplasty. Zywiell MG, Daley JA, Delanois RE, Naziri Q, Johnson AJ, Mont MA. Int Orthop. 2011 Jul;35(7):1001-6.
10. A Randomized, Clinical Trial of Preadmission Chlorhexidine Skin Preparation for Lower Extremity Total Joint Arthroplasty. Kapadia BH, Elmallah RK, Mont MA. J Arthroplasty. 2016 Dec;31(12):2856-2861
11. Effectiveness of various hospital-based solutions against community- acquired methicillin-resistant Staphylococcus aureus. Perona PJ, Johnson AJ, Perona JP, Issa K, Kapadia BH, Bonutti PM, Mont MA. J Long Term Eff Med Implants. 2013;23(1):23-9.

Therefore, one can already begin to appreciate the importance of skin decontamination in the prevention of PJIs. The majority of PJIs occurring within 1 year of surgery are initiated through the introduction of microorganisms at the time of surgery through the skin. In fact, the most common causative bacterial organisms, Staphylococcal species, are common skin flora.

The impact of ventilation in operating rooms in the control of infections has been the subject of a great deal of research. There was a time when it was commonly thought that airborne contamination of the operating surgical wound was a major source of infection. Thus, strategies were developed to make the air of ORs, especially those used for joint arthroplasties, as clean as possible. This led to such therapies as the use of laminar flow, systems that blow high velocity HEPA-filtered air around the OR table, as well as “space suits” to exhaust air from the surgical staff outside the surgical field. Recent studies have shown these strategies may be ineffective and indeed potentially harmful. In the United States, the current standard for ORs calls for maintaining positive pressure with at least a minimum number of air exchanges per hour. In addition, filtered air enters the OR generally from the ceiling in a downward manner and is exhausted out the sides of the OR. The levels of filtration and the specifics of the HVAC standards are established by ASHRAE, the American Society of Heating, Refrigerating and Air

Conditioning Engineers. It is my understanding that details concerning OR ventilation and filtration, as well as ASHRAE Standards, are being addressed by other experts. For purposes of my opinions from the standpoint of an orthopaedic surgeon, it is clear that: a) airborne transmission of bacteria plays a very minor role in the formation of PJIs; b) use of such strategies as laminar flow and “space suits” have not proven to be effective at reducing PJIs and, in fact, appear to increase the risk of PJIs; and c) the standard ventilation system in the ORs in the U.S. is turbulent, not laminar. Experiments, cited by Plaintiffs’ experts, are inappropriately being used to imply that use of the Bair Hugger disrupts turbulent air flow systems. However, turbulent air systems are not sensitive to airflow disruption in the manner purportedly demonstrated in these experiments involving laminar flow. Therefore, these experiments are irrelevant to the issue of whether the Bair Hugger can cause or increase the risk of PJIs.

A related issue raised by plaintiffs is the number of bacteria necessary to cause a deep periprosthetic joint infection. It is likely that the number of bacteria necessary to inoculate a joint implant sufficient to cause a PJI is less than for a superficial infection, but certainly far more than 1 as has been argued by Plaintiffs’ experts. An important factor is the presence of foreign material (prosthesis) that does not have a blood supply, and the fact that the host may be less able to ward off an infection as well as it can for the skin where there is a blood supply and more host immune mechanisms present. However, despite this, large numbers of bacteria are still needed for an infection. For example, close to 100 colony forming units (CFUs) of *S. aureus* are necessary to establish infection if inoculated at the time of a hip hemiarthroplasty in a rabbit model, compared with 10^4 when no implant is placed. This difference is explained by biofilm formation in the case of the foreign body (Southwood RT, Rice JL, McDonald PJ, Hakendorf PH, Rozenbils MA. 1985. Infection in experimental hip arthroplasties. J. Bone Joint Surg. Br. 67:229–231). To further answer this question, it is important to understand that

bacteria must reproduce in sufficient numbers to overwhelm the host immune response. The metric to describe this is the Infectious Dose or ID₅₀ for that bacteria, which is the number of bacteria required to cause an infection in 50% of exposed hosts. The number of bacteria needed to cause an infection also depends on the host. Patients with normal immune systems are more difficult to infect than immunocompromised patients. The ID₅₀ for most bacterial organisms that cause SSIs and PJIs can range from 10³ and 10⁶, and often greater (millions of cells).

While animal models and biologic plausibility do suggest that the amount of bacteria necessary to cause a PJI is less than that needed to cause an SSI, there are no studies that suggest that the number is as low as 1 or 2 CFUs. The animal model noted above that showed inoculation with 100 CFUs is the lowest number in any animal study. There is no evidence to suggest the idea that fewer CFUs could cause PJIs in humans, and it is likely higher than that.

Particles are not the same as bacteria. Only a small minority of particles carry bacteria and many bacteria may not be viable (alive) when they actually come in contact with the host. Particles, which can be easily measured in real time (as opposed to bacteria, which require sampling and culturing), are commonly used as a surrogate to get a crude “snapshot” of what might be the bioburden in an OR. If particle counts are low, that is an indicator that viable bacterial counts are also low. If particle counts are high, that may suggest that the bacterial burden could also be high, but that is not necessarily the case. Studies have demonstrated that particles are, in general, a poor-to-mediocre surrogate for bacterial measurements.

Plaintiffs’ experts rely on experiments conducted by employees and agents of the maker of a competitive warming device that purport to show that use of the Bair Hugger can increase particle counts. In the absence of any other information, these findings would warrant further investigation to see if the demonstrated increase in particle counts correlates with an increase in bacteria. However, there are now nine published studies that have examined that question and

found no increase in bacteria from Bair Hugger use. In addition, researchers connected to the maker of the competitive warming device tried **on at least seven occasions** to demonstrate that use of the Bair Hugger increased bacteria. These efforts were also unsuccessful and, importantly, were never published. These same researchers published the results of their experiments showing increases in particle counts - the implication of these particle studies was that the increased particles correlated with increased bacteria, which these researchers already knew was not the case (and failed to disclose). Plaintiffs' experts rely on the assertion that particles are a valid surrogate for bacteria generally (which, as discussed above, is itself an inaccurate proposition) and then make the unsupported assumption that, because a handful of experiments demonstrated that the Bair Hugger could, under certain experimental conditions, increase particle counts, then it must also be increasing bacteria. Plaintiffs' experts make this leap without regard to the weakness in the claim that particles can be a valid surrogate for bacteria in general, and without regard to the **16** consistent studies that demonstrate that the Bair Hugger device, regardless of its impact on particle counts, does not increase the bacterial bioburden. Bacteria cause PJIs, not particles. Plaintiffs' efforts to take the weak and controversial evidence that particles may be used as surrogates for bacteria, combine that with limited experimental evidence that the Bair Hugger may increase particles, and then conclude that the Bair Hugger does in fact increase bacteria, in the face of at least 16 studies to show that it does not, is pure sophistry and does not comport with valid scientific methodology.

The multiple studies that show no increase in bacteria with the Bair Hugger device include 9 published and **7** non-published. The following **7** non-published studies are confirmed by resources connected to Dr. Augustine, the maker of a competitive warming device: 1) an effort by McGovern and Reed to culture bacteria; 2) an effort by Legg to culture bacteria; and **3**

five attempts by Albrecht, working for Augustine, to culture bacteria. None were successful and none were published.

The published studies that have looked for and failed to find an increased bacterial bioburden associated with Bair Hugger include the following:

Hall poster 1991
Zink 1993
Dirkes 1994
Avidan 1997
Tumia 2002
Huang 2003
Moretti 2009
Occhipinti 2013
Oguz 2017

Many things in the operating room impact airflow. It was claimed by Plaintiffs' experts that the Bair Hugger can impact airflow in the operating room. In my opinion, even if this was the case, it would be infinitesimal in comparison to so many other sources of airflow generation in the operating room, which include:

1. Surgeon traffic - he or she is performing the procedure and creating continual air-currents directly at the operative site
2. Surgical assistants - the same can be said for the 1 to 3 assistants helping in any lower extremity arthroplasty procedure
3. Nurse or Surgical techs - handing instruments and other measures to help with the procedure
4. Circulating nurses - handing out instruments, prostheses, etc.
5. Other members of operating room team - anesthesiologists, others that enter room, delivering of blood, etc.
6. Doors opening and shutting creating wind currents
7. Moving of lights and other equipment directly creates waves or currents by individual (surgeon or team), as well as the specific object moving
8. Many pieces of equipment in the OR generate air currents, including those that have cooling fans.

The airflow generated by the Bair Hugger, as it emerges from the multiple perforations in the warming blanket, is very gentle. Moreover, the Bair Hugger is placed such that the air blows directly on the patient, underneath multiple drapes, and any airflow that emerges from under the drapes is so low in velocity that it has no impact on the air currents in an OR. This is especially true when one considers all of the other sources of air movement during surgery.

In summary, any current created by the Bair Hugger would be negligible compared to these other sources, and therefore, should be considered non-existent.

There are many sources of heat generation in the operating room that are far in excess of any heat generated from the Bair Hugger device. For example, 4 people involved in the operating room, as well as being much closer to the operative site than a Forced Air Warmer, generate much more heat than the Forced Air Warmer, and there are many more heat sources closer to the field. Heat from the Forced Air Warmer is further away from the field and would be dissipated by approximately the inverse square ($1/r^2$) of the distance, so once again, it would have negligible to no effect.

The many sources of heat in the operating room of lower extremity joint arthroplasties include:

1. Saw blades on bone - with the bone generating heat
2. Batteries that power the saw blades as they are used
3. Many surgeons use hooded gowns with battery packs (space suits), and air is blown inside these suits
4. General overhead lights in any operating room
5. Focused overhead lights directly at field (usually 2 of them)
6. Ancillary hooded lights that many surgeons wear (and the light generating unit)
7. All personnel in operating room, including:
 - a. Patient

- b. Operating surgeon and direct assistants (often 2 to 4)
 - c. Anesthesiology team-often 2
 - d. Circulating nurses, preparation help, assistants (often 2)
8. Machine to process fluid irrigation fluids - vacuum canisters and more substantial canisters used nowadays that generate much heat
 9. Often other power sources for special blades used in some surgeries (more often revisions) for burring bone, cement, etc - Anspach/Midas Rex devices generate a tremendous amount of heat
 10. Standard electrocautery devices
 11. Ancillary cautery devices - Plasmablate, Aquamantis, Canady, and others
 12. Various ancillary devices in the operating room by anesthesiologist, e.g. defibrillator, computer, their monitor, etc; their anesthesia machine is a source of heat

Finally, the Forced Air Warmer, which is away from the operative field, and has a negligible effect compared to many of the devices or other sources of heat generation mentioned above. See Exhibit D attached hereto.

HEPA filters do not reduce the bacteria that cause surgical site infections. Plaintiffs argue that the Bair Hugger is defective because it fails to use a HEPA filter and, as a result, the Bair Hugger causes more PJIs than if it had a HEPA filter. This opinion is contradicted by both the microbiology and a recent study demonstrating that a HEPA filter does not reduce PJIs.

A HEPA filter, or high efficiency particle arresting filter, is designed to capture 99.97% of particles the size of .3 microns, which is considered the most penetrating particle size, or MPPS. Particles larger and smaller than .3 microns are captured at a higher rate. Bacteria that cause PJIs are actually much larger than .3 microns. An individual bacterium of the kinds that cause PJIs typically ranges from .7 to 1.2 microns, and these bacteria generally travel in clusters (thus increasing their size) and, if traveling by airborne route, generally travel on fomites that are even larger.

Thus, the HEPA's high capture rate of .3 microns particles is not a relevant consideration for bacteria. The question is what is adequate to capture PJI-causing bacteria? ASHRAE standards call for OR ventilation systems to have filters with a MERV rating of 14 for general surgery. A MERV-14 filter is highly efficient at capturing PJI-causing bacteria. Note: the ASHRAE standards are applicable to the ventilation system, not pieces of equipment used in OR, virtually none of which have filters of any kind.

The Bair Hugger, in fact, has a filter that is rated MERV-14, and if the Bair Hugger were a ventilation system, it would meet ASHRAE standards. The fact that it has its own MERV-14 is, in effect, icing on the cake in that it is filtering air that has already been filtered through the hospital's HVAC system. Because the air from the Bair Hugger is so far removed from the surgical site, as discussed above, it is questionable whether it needs a filter at all. Nevertheless, the MERV-14 filter incorporated in the Bair Hugger is far more than adequate to capture PJI-producing bacteria. A recent study conducted by colleagues of mine at the Cleveland Clinic has, in fact, demonstrated that a HEPA filter does not reduce PJIs when incorporated in a forced air device.

At the Cleveland Clinic, approximately 2 years ago, a switch was made from the Bair Hugger device to the Mistral-Air Forced Air Warming System (Stryker, Portage, Michigan). This forced air warming system included a premium HEPA air filtration system. With a hospital system that performs over 4,000 lower extremity joint arthroplasties per year, this afforded a unique opportunity to compare infection rates with both devices. The abstract for the study has been accepted by the MusculoSkeletal Infection Society (MSIS) and will be presented in August of this year at the MSIS annual meeting. A copy of the abstract to be presented at the MSIS meeting is attached hereto as Exhibit E. Because the MSIS requests that abstracts not be publicly disclosed prior to presentation, I have designated this abstract as confidential. This

designation will no longer be necessary after the MSIS meeting ends on August 5. Of note, the study found that there was no statistically significant difference in infection rates between the Bair Hugger with a MERV-14 filter and the Mistral Air with a HEPA filter. I further note that, with respect to PJIs, the study demonstrated that the infection rate was actually lower with the Bair Hugger than with the Mistral Air, 0.47% vs. 0.77%; however, this difference was not statistically significant ($p=0.15$).

Based on the foregoing, it is my opinion that a HEPA filter is not necessary for the Bair Hugger, nor would a HEPA filter on the Bair Hugger have any positive impact on PJI rates. It is my further opinion that the Bair Hugger is not defectively designed as a result of it not having a HEPA filter and that the MERV-14 filter it has is more than adequate for the device. The results show no statistical differences in infection rates between the two devices. The clear finding of this study is that the HEPA system does not influence infection rates.

Odds ratio of 3.8 for an infection using the Bair Hugger device in the McGovern, et al. study is fallacious for multiple reasons. The study by McGovern is cited often in the Plaintiffs' expert reports as proof of the Bair Hugger device leading to increased PJIs (Forced-air warming and ultra-clean ventilation do not mix: an investigation of theatre ventilation, patient warming and joint replacement infection in orthopaedics. J Bone Joint Surg Br. 2011 Nov;93(11):1537-44. McGovern PD, Albrecht, Belani KG, Nachtsheim C, Partington PF, Carluke I, Reed MR).

The reduction in infection rates shown in the McGovern paper can be explained by the Hawthorne effect, regression to the mean, and most importantly by multiple real confounding factors. The "Hawthorne effect," operated because an educational program was introduced to the entire staff in an effort to reduce infection rates. When individuals are simply being observed, they will perform differently — wash hands more often, take more care in what they

do, and engage in other improved forms of “aseptic technique.” These actions will invariably lead to reductions in PJI rates. In addition, the high infection rates during the early part of the study period would also most certainly have experienced a regression to the mean and been reduced. However, what is most important to understand is that there were a multitude of not only hypothetical, but real confounding factors that led to the high reported rates of the Bair Hugger device when compared to the conductive fabric device. During some time periods, infection rates were in fact lower for the Bair Hugger. The following will elaborate on these confounding factors:

- a. When one is using a single factor analysis, any conclusions in any report can easily be biased by multiple confounding factors that clearly exist in this report. A multiple regression analysis of variance should have been used for appropriate scientific evidence, and in my opinion, the authors reached erroneous conclusions about the effects of the Bair Hugger device on PJIs. In a true highest level prospective randomized study, one would compare two groups that were matched for as many relevant variables as possible.
- b. The conductive fabric device was not the only change made at the time that the Bair Hugger was discontinued, but rather multiple other practices had been implemented, any or all of which could have influenced infection rates: more surveillance (hiring of two dedicated SSI nurses), better infection-control techniques, lowering operating room traffic (as mentioned in #1 above), footwear changes, change to more effective antibacterial wound dressings, no shaving of surgical sites (increases infection rates when performed), screening for methicillin-susceptible staphylococcus aureus (MSSA), prewarming, and switch to chlorhexidine wound preps.

- c. A number of infected cases were counted in the Bair Hugger group before the general surveillance methods had been introduced. This obviously favored the conductive fabric group in the analysis.
- d. From testimony, infections may have been placed erroneously in the wrong group — erroneously increasing the infection rate for the Bair Hugger group and decreasing the infection rate for the conductive fabric group.
- e. There was a notable switch in deep venous thrombosis (DVT) prophylaxis during the time periods studied. Some agents can cause increased bleeding — more hematomas occurred in this study, and this can lead to infections. This would have been a major confounding factor in the study and was particularly interesting to me as the primary author of the American guidelines for DVT prophylaxis, as reflected in the following references:

1. Preventing venous thromboembolic disease in patients undergoing elective hip and knee arthroplasty. Mont MA, Jacobs JJ, Boggio LN, Bozic KJ, Della Valle CJ, Goodman SB, Lewis CG, Yates AJ Jr, Watters WC 3rd, Turkelson CM, Wies JL, Donnelly P, Patel N, Sluka P; AAOS. J Am Acad Orthop Surg. 2011 Dec;19(12):768-76.

2. American Academy of Orthopaedic Surgeons clinical practice guideline on: preventing venous thromboembolic disease in patients undergoing elective hip and knee arthroplasty. Jacobs JJ, Mont MA, Bozic KJ, Della Valle CJ, Goodman SB, Lewis CG, Yates AC Jr, Boggio LN, Watters WC 3rd, Turkelson CM, Wies JL, Sluka P, Hitchcock K. J Bone Joint Surg Am. 2012 Apr 18;94(8):746-

3. Preventing venous thromboembolic disease in patients undergoing elective total hip and knee arthroplasty. Members of 2007 and 2011 AAOS Guideline Development Work Groups on PE/VTED Prophylaxis., Mont M, Jacobs J, Lieberman J, Parvizi J, Lachiewicz P, Johanson N, Watters W. J Bone Joint Surg Am. 2012 Apr 18;94(8):673-4.

In the Jensen study on the same patient population, published approximately one year before the McGovern et al. report, the infection rate was found to be higher in the short-lived rivaroxaban period. Although it did not reach statistical significance (5 of 489 patients vs. 14 of 559 patients, 1% vs. 2.5%, $p = 0.102$), it did prompt a switch. Statistics

are unimportant here. The work is underpowered and no hospital would allow 14 infections (almost three times the rate of the other cohort) to not be addressed. Moreover, it is my understanding that Prof. Holford conducted a re-analysis of the Jensen study using the same inclusion and endpoint criteria used in McGovern and demonstrated that there was a statistically significant impact from rivaroxaban. In fact, it was addressed when noted before switching back to the previously used drug, and therefore is a major confounding factor.

- f. Likewise, there was a shift in antibiotic use that certainly could have influenced the infection rates. During a 5 months period of Bair Hugger device use compared to 7 months of conductive fabric use with identical antibiotics and DVT prophylaxis, there were no differences in infection rates.
- g. In addition, another unusual aspect of this study is that there was a significantly greater infection rate for hips when compared to knees – these should be roughly equal. This finding calls into question the infection rates at this institution, and suggests the possibility of an aberrant hospital-wide or surgeon-specific issue with surgical technique in hip arthroplasties. If a single surgeon who performs mostly hip procedures is using sub-par technique, this could explain the unusual ratio with hip to knee infections

In summary, when parallel patient populations were compared, they were not statistically different. This is why for the vast majority of the time period, one is not comparing apples to apples, but rather apples to so many different factors, which makes conclusions about the Bair Hugger device from a single observational study completely erroneous. One could have picked any one of the 20 other factors that were changed and reached the same conclusions about that particular factor.

Warming or hypothermia does in fact decrease SSIs and maintenance of normothermia has multiple beneficial effects. Strong evidence of SSI reduction for active warming was found by Kurz (1996) and Melling (2001). Since these two seminal studies, warming has become the standard of care and it would not be possible to obtain IRB approval to conduct a randomized study comparing warming to no warming. Thus, more recent studies have been conducted in other ways and have examined other endpoints as well as infection. The well-established body of medical literature demonstrates that maintaining normothermia in surgical patients results in many benefits including reduced blood loss, reduced need for blood transfusions, reduced cardiac incidents, reduced anesthesia recovery time and time in PACU, reduced pain and need for pain medication, reduced shivering, increased patient comfort, reduced hospital length of stay, and reduced mortality. Moreover, studies continue to demonstrate the importance of maintaining normothermia as part of an infection prevention strategy. As the rates of infection have gone down due to multiple strategies and improved surgical techniques, the ability to demonstrate a significant impact of normothermia on infection rates is more difficult. Nevertheless, it is well accepted that warming reduces infection rates in all surgical categories including orthopaedics. Warming remains the standard of care and is recommended by all medical standards groups.

As one recent paper has noted:

Maintenance of normothermia in orthopedic surgery has proven to have broad implications from bench top to bedside. Normothermia has been shown to impact everything from nitrogen loss and catabolism after hip fracture to infection rates after elective arthroplasty.

Allen & Jacofsky, Normothermia in Arthroplasty, the Journal of Arthroplasty (2012).

Sterile vs. aseptic concept of the operating room environment: The operating room is not a sterile hood for microbiological experiments. We are simply trying to effectuate a reduction of bacteria which are ever-present. “Dilution is the solution to pollution.” We can’t

eliminate every bacteria — the only things sterile are the implants and the instruments — but only briefly. One cannot sterilize skin, particularly the lower layers.

When one preps the patient for a total knee or hip arthroplasty, there is a tremendous amount of draping that occurs that isolates the wound from the rest of the operating room. This applies importantly to the anesthesia team, their equipment, and the proximal end of the body, where the Bair Hugger device sits. This prepping for surgery can be extensively illustrated to show how there can be little to no contamination from objects at the front of the operating room table. See Exhibit F, attached hereto.

There are a myriad of potential sources of bacteria in the OR. Besides the patient and the staff, equipment can become contaminated during surgery (e.g., electrocautery tips, drills, other surgical instruments); even with double-gloving, inadvertent nicks of the surgeon's gloves are common, (and often undetected until after surgery); overhead lights, etc. See Exhibit G, attached hereto. The goal of aseptic techniques is not the elimination of every single bacterium, an impossible task; rather, it is the reduction of bacteria to the greatest extent possible without compromising the efficacy of the surgery. Thus, aseptic practices speak in terms of “log reductions” in bacteria counts, not sterilization. This is an important concept to remember when assessing any theoretical impact that a device such as the Bair Hugger could have on the bacterial burden in the operative field.

Variations in skill of surgeon or surgical technique can markedly influence infection rates. Many surgeons will perform 5 per month vs. 50 to 100. Also, there can be tremendous variations by institution. This can have a major impact on PJI rates.

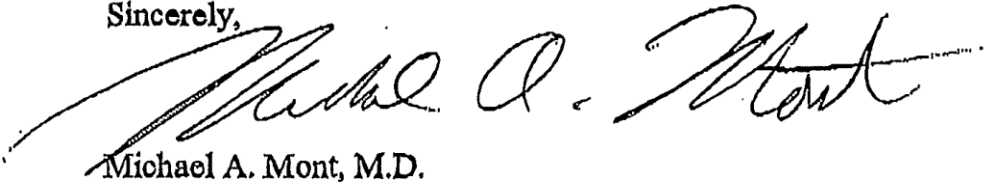
SUMMARY

It is my opinion that the Bair Hugger is not defectively designed. It is safe and effective at maintaining normothermia, which in turn confers a wide range of benefits on the patient,

including reduction of infection risk. Further, use of the Bair Hugger does not cause PJIs nor does it contribute in any way (let alone in a substantial way) to the risk of developing a PJI.

All of the above opinions are held to a reasonable degree of medical certainty. I reserve the right to supplement this report should I receive any additional information relevant to my opinions.

Sincerely,

A handwritten signature in black ink, appearing to read "Michael A. Mont". The signature is fluid and cursive, with a long horizontal stroke extending to the right.

Michael A. Mont, M.D.

June 2, 2017